



Clinical Communications: Adult



A CASE OF HEATSTROKE COMPLICATED BY PERSISTENT VENTRICULAR TACHYCARDIA

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Abstract—Background: Patients suffering from heatstroke often present with electrocardiograph abnormalities, but persistent ventricular tachycardia has not been reported previously. **Objective:** This report is intended to demonstrate that rapid and effective cooling is critical to survival in patients experiencing heatstroke with ventricular tachycardia. **Case:** We cared for a healthy 38-year-old man with environmentally induced heatstroke, who presented with ventricular tachycardia resistant to cardioversion until his core body temperature was reduced significantly. **Conclusions:** This case represents the first report of ventricular tachycardia occurring in a patient with heatstroke. Successful cardioversion could not be achieved until his core body temperature was lowered significantly, reinforcing the need for rapid temperature reduction that can be accomplished through noninvasive means. © 2015 Elsevier Inc.

Keywords—heatstroke; heat stroke; hyperthermia; ventricular tachycardia; temperature reduction

INTRODUCTION

Heatstroke is characterized by hyperthermia to temperatures $> 40^{\circ}\text{C}$ with associated altered mental status. It is considered an acute life-threatening emergency and carries a mortality rate as high as 30% to 80% (1–4). The hyperthermia results from the body's inability to regulate heat production and dissipation. Heatstroke typically occurs in individuals exposed to high outdoor temperatures in combination with the ingestion of

prescribed or illicit medications, such as antimuscarinics or sympathomimetics (4,5).

Persistent ventricular tachycardia typically occurs in patients with underlying structural heart disease, coronary artery disease, or in patients taking dysrhythmogenic medications. Although electrocardiogram (ECG) abnormalities are commonly seen in patients with heatstroke, we found no reports of persistent ventricular tachycardia in conjunction with heatstroke presentation in the literature (6). In our patient, temperature-sensitive ventricular tachycardia developed during environmentally induced heatstroke, despite the lack of underlying heart disease or the ingestion of prescribed or illicit medications. Successful electrical cardioversion was only accomplished after the lowering of his core temperature through the use of multiple cooling methods.

CASE REPORT

A previously healthy 38-year-old male construction worker clothed in jeans and a long-sleeve shirt was found unresponsive at his work site on a hot summer day, where ambient temperatures were recorded at 92°F (33.3°C). Paramedics were called and found him actively convulsing, but his tonic-clonic activity ceased spontaneously. Before arrival at our facility, he received 500 mL normal saline intravenously, dextrose 25 g, sodium bicarbonate 50 mEq, and thiamine 100 mg. Ice packs were placed in the axilla and groin and his initial vital signs revealed a systolic

blood pressure of 80 mm Hg, pulse of 176 beats/min (bpm), and respiratory rate of 24 breaths/min.

On arrival to the emergency department, 35 min after the paramedics first reached the patient, his blood pressure was 109/36 mm Hg, pulse was 162 bpm, respiratory rate was 20 breaths/min, with an oxygen saturation of 99% on a nonrebreather mask. His core temperature, obtained from a rectal thermometer, was 108°F (42.2°C), which was the maximum for that temperature probe. The patient was unresponsive but moving all four extremities, pupils were 1 mm bilaterally, skin was very warm and dry, and no rash was evident. No family or friends accompanied the patient and no medical alert bracelet or pill bottles were found.

The patient was placed on cardiac monitoring that demonstrated ventricular tachycardia at a rate of 160 bpm (Figure 1). Synchronized cardioversion was attempted at 200 J with no change in rhythm. Naloxone 0.4 mg was administered intravenously without change in mental status and the patient was intubated for airway protection after pretreatment with etomidate 20 mg and rocuronium 50 mg. During the resuscitation, the patient received amiodarone 300 mg, sodium bicarbonate 50 mEq, lidocaine 100 mg, magnesium 1 g, calcium gluconate 1 g, and lorazepam 2 mg. Synchronized cardioversion was repeated five times over the next 30 min at 360 J without effect.

Simultaneous with the resuscitation, evaporative cooling using a large fan and the spraying of room-temperature water was started and fresh ice packs were placed in the groin and axilla. In addition, a nasogastric

tube and a three-way Foley catheter were inserted with continuous irrigation of ice-cold water and a cooling blanket was placed under the patient. A continuous rectal probe was inserted to monitor the resuscitation. Approximately 23 min after aggressive cooling was begun, his temperature was 109.5°F (43°C).

When the patient's temperature had fallen to 106°F (41.1°C) 42 min into the resuscitation, synchronized cardioversion was again attempted with successful conversion to sinus tachycardia. After cardioversion, his blood pressure was 103/40 mm Hg and an amiodarone infusion was started. By 73 min, the patient's temperature had fallen to 100.5°F (see Table 1), he was noted to be moving all extremities and a versed infusion was started for sedation. Antibiotics consisting of ceftriaxone 2 g, Vancomycin 1 g, Zosyn 4.5 g, and Amikacin 1 g were administered for broad coverage of possible infectious etiology. Lastly, he was also given phenytoin 1 g for a possible epileptic origin of his seizure.

Computed tomography of the head and plain radiography of the chest were both normal. Arterial blood gas on arrival revealed a pH of 7.28, bicarbonate of 18 mmol/L, and lactic acid of 10.4 mmol/L. Serum chemistries were as follows: sodium 127 mmol/L, potassium 6.1 mmol/L, chloride 94 mmol/L, blood urea nitrogen (BUN) 24 mg/dL, creatinine 2.3 mg/dL with an anion gap of 15. Creatine kinase was elevated at 4033 IU/L. A urine test for drugs of abuse was negative.

The patient was transferred to the intensive care unit, where he developed multisystem organ failure, manifest

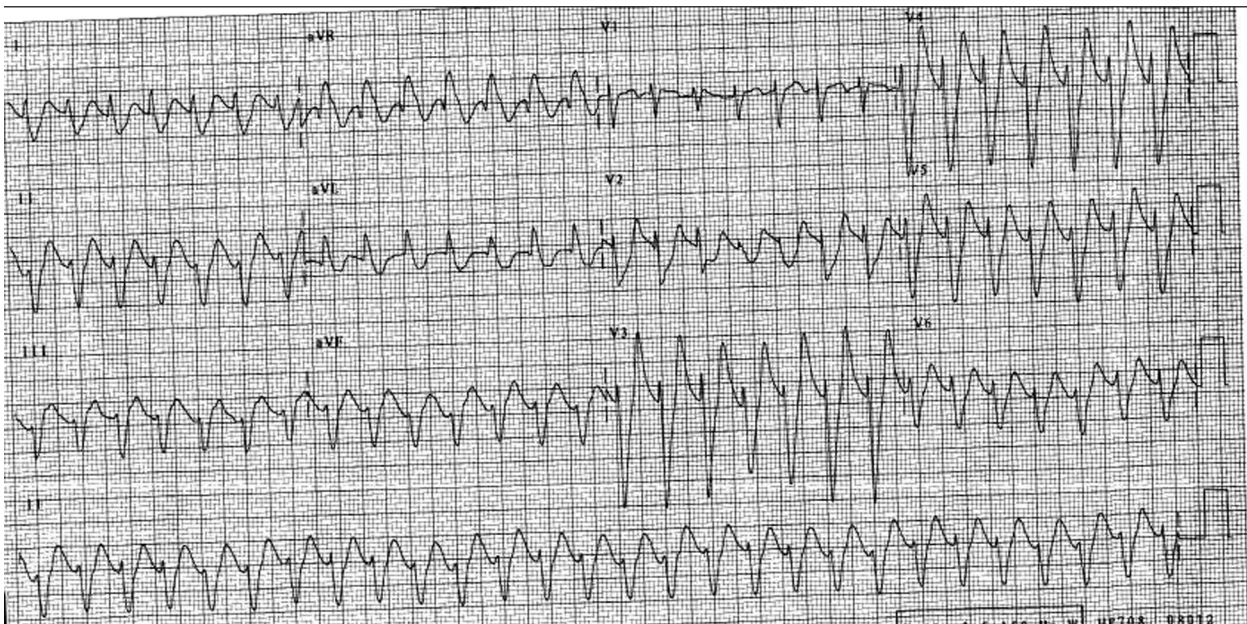


Figure 1. EKG of patient on arrival.

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